

Additional studies suggest that even smaller reductions in sodium intake over an extended period may reduce blood pressure, as discussed below. In a 2-year controlled trial, 31 hypertensive patients were instructed to reduce sodium intake by about 70 to 100 mEq. Although sodium excretion declined only from 191 to 157 mEq, indicating a much smaller reduction in intake, the average diastolic blood pressure fell by 7.3 mm Hg, a result similar to that obtained by drug treatment (Morgan et al. 1978).

Similar results have been obtained in even shorter time periods. In a 12-week randomized trial, one-half of 90 drug-treated hypertensive patients were placed on a no-added-sodium diet (estimated to be equivalent to a maximum intake of about 100 to 150 mEq, or 2,300 to 3,450 mg of sodium). Among the salt-restricted patients, sodium excretion decreased from 150 to 37 mEq and diastolic blood pressure decreased by 6.3 mm Hg (compared with a fall of 2.9 mm Hg in the drug-treated control group), and four of five patients were able to discontinue or reduce medication (Beard et al. 1982).

In a randomized 8-week crossover study of 19 hypertensive patients, dietary restriction was associated with a reduction in sodium excretion from 191 to 83 mEq and a fall in mean blood pressure of 7.6 mm Hg (MacGregor, Markandu, et al. 1982). During a later double-blind phase of the trial, a level of sodium excretion of 76 mEq was associated with a mean blood pressure of 7.1 mm Hg lower than that of the control group (MacGregor, Smith, et al. 1982). Results of a nearly identical trial, however, showed no difference in patients' blood pressures (Watt et al. 1983). The differences between these results may be related to a somewhat lower blood pressure at entry, to a slight difference in average sodium excretion, or to chance variation.

A recent review of 13 randomized clinical trials concluded that moderate (37 to 153 mEq/day) sodium restriction does reduce blood pressure, at least to some small extent, and that this practice is most effective for systolic pressures, for older hypertensive patients, and for patients with higher initial blood pressures (Grobbee and Hofman 1986a).

Prevention

The important question of whether reduction in sodium intake might prevent the rise in blood pressure with age has been addressed in a few short-term studies of adults, children, and infants with normal blood pressures (normotensives). Blood pressure response to moderate sodium restriction (to 60 mEq) for 3 months in 16 healthy normotensive husbands and wives was associated with a decrease in sodium excretion from 152.7 to 59.5 mEq/day and significant decreases in systolic and diastolic blood

pressure of 4 and 3 mm Hg, respectively (Miller et al. 1983). In a randomized double-blind trial, 231 infants fed a diet reduced in sodium content by two-thirds during the first 6 months of life had a mean systolic blood pressure that was significantly lower (2.1 mm Hg) than that of 245 infants fed a customary diet (Hofman, Hazebrock, and Valkenburg 1985). These two studies indicate that apparently normal blood pressures can be reduced by dietary sodium restriction, and they suggest that this practice might prevent age-related increases in blood pressure. To date, however, no reported studies have tested this hypothesis directly.

Variability in Response

The disparate blood pressure responses to sodium intake in animals attributed to genetic influence (Dahl, Heine, and Tassinari 1962) have led to the proposal that dietary sodium and blood pressure relationships are controlled by similar genetic factors in humans (Dahl 1972). Support for the concept of individual variability in human blood pressure response has been provided by a study of 20 hypertensive patients in hospital metabolic units who were fed daily sodium intakes of 9 mEq, 109 mEq, and 249 mEq. Investigators were able to distinguish a salt-sensitive group, whose blood pressures fell in response to sodium restriction, and a salt-resistant group, whose blood pressures tended not to be influenced by changes in salt intake (Kawasaki et al. 1978). It has not yet been possible, however, to identify a way to consistently distinguish salt-sensitive from salt-resistant persons other than by measuring the blood pressure response itself.

While acknowledging that there are some hypertensive individuals whose blood pressures do not respond to sodium restriction, some investigators believe that patients who continue to consume low-salt diets have a more benign course than those who revert to a high salt intake (Dahl 1977).

Two dietary intervention studies suggest that weight loss or sodium restriction or both can retard the return of high blood pressure in patients who have achieved normal blood pressure levels through drug treatment. The Dietary Intervention Study in Hypertension assessed whether hypertensive patients whose hypertension was controlled with drugs could discontinue these medications with or without dietary therapy. The 415 patients who discontinued medications were randomized, stratified by body weight, either to control, weight loss (mean loss of 10 lb), or sodium restriction (mean decrease of 40 mEq/day). The patients in the dietary intervention groups were more than twice as successful as those in the control group in maintaining normal blood pressure. At the end of 56 weeks, 50 percent of these patients remained normotensive without medication. This success rate was higher for patients with pretreatment mild

hypertension than for those with pretreatment severe hypertension. In the former group, 71.5 percent in the weight loss group and 78 percent in the nonobese sodium restriction group remained normotensive (Langford et al. 1985).

Similar results were noted in a 4-year trial, the Hypertension Control Program. Among patients with pretreatment mild hypertension who discontinued antihypertensive medications but who restricted calories and sodium and alcohol intake, 39 percent maintained normal blood pressure after 4 years. In contrast, only 4 percent of those patients who did not receive nutritional intervention remained normotensive (Stamler et al. 1987).

It should be noted that in the two studies previously described, the patients had relatively mild hypertension that had been controlled successfully with drugs for the preceding 5 years. Although the long-term effectiveness of dietary intervention in persons with more severe elevations in blood pressure remains to be established, these studies have important implications for the millions of Americans who are being treated for high blood pressure or who have elevated diastolic blood pressure. Many persons who adhere to a dietary regimen to control blood pressure can reduce or stop drug therapy, with potential economic and health advantages.

Role of Alcohol in Hypertension

Epidemiologic studies have shown that individuals who regularly consume large amounts of alcohol have higher blood pressure than people who abstain from alcohol or who drink only moderate amounts. The epidemiologic literature for the existence and nature of an association between blood pressure and alcohol consumption has been reviewed (MacMahon 1987). In the 12 cross-sectional North American studies reviewed (Table 3-6), with the exception of the Canada Health Study, all reports showed a significant positive association that was independent of age, relative body weight, exercise, and smoking status. In most U.S. studies, a J-shaped or U-shaped association is observed between blood pressure and alcohol consumption, with blood pressure greater in nondrinkers than in those consuming one to two drinks per day. It is not clear whether this might be partially explained by other factors, such as obesity. A more consistent finding was elevation of blood pressure at levels of three drinks or more per day. The prospective, observational studies of the association of blood pressure with alcohol consumption (Table 3-7) have also been reviewed (MacMahon 1987). In this group of investigators, only the Honolulu Heart Study failed to show a positive association with either systolic or

Table 3-6
Studies of the Cross-Sectional Association of
Blood Pressure With Alcohol Consumption

Year	Study ^a	No. of Subjects	Male Subjects (%)	Age (years)
<i>North American</i>				
1967	Los Angeles Heart	865	100	21+
1977	Chicago W. Electric	1,899	100	40-55
1977	Kaiser-Permanente I	83,947	45	15-79
1980	Tecumseh	3,390	47	18+
1981	Lipid Research Clinics	4,783	52	20+
1981	Honolulu Heart	8,006	100	46-68
1983	Stanford Five City	1,842	48	20-74
1983	Framingham	5,209	42	29-62
1985	Canada Health	1,418	51	20+
1985	NHANES	9,553	45	18-74
1986	Kaiser-Permanente II	66,510	44	—
1986	Albany	1,910	100	38-55
<i>European</i>				
1974	Copenhagen	5,249	100	40-59
1980	Yugoslavia	11,121	100	35-62
1982	Lyon	1,134	100	20-59
1983	North Karelia/Kuopio	8,479	50	30-64
1984	Munich Blood Pressure	3,198	33	30-69
1985	Wurttemberg	3,351	88	20-65
1985	Zutphen	794	100	40-59
<i>Australian and New Zealand</i>				
1980	Tasmania	85	100	36 ^b
1981	CSIRO	350	100	23 ^b
1982	Sydney Hospital	20,920	65	18-70
1982	Perth	491	100	20-45
1984	Medicheck	11,000	75	43 ^b
1984	Australian RFPS	5,550	50	19+
1985	Milton	901	56	19+
1985	Auckland	1,429	66	35-64
<i>Japanese</i>				
1984	Osaka/Akita	887	100	40-69
1984	Minamikawachi	3,083	37	53 ^b

^aCSIRO = Commonwealth Scientific and Industrial Research Organization; NHANES = National Health and Nutrition Examination Survey; RFPS = Risk Factor Prevalence Study.

^bMean age.

Source: Adapted from MacMahon 1987.

Table 3-7
Prospective Observational Studies of the Association of
Blood Pressure With Alcohol Consumption

Year	Study	No. of Subjects	Male Subjects (%)	Age (years)	Followup (years)
1977	Chicago People's Gas	1,340	100	40-59	4
1981	Chicago W. Electric	871	100	40-55	4
1982	Honolulu Heart	6,858	100	46-68	6
1983	Framingham	5,209	42	29-62	4
1985	Zutphen	794	100	40-59	10
1986	Albany	1,910	100	38-55	18

Source: Adapted from MacMahon 1987.

diastolic blood pressure at followup with alcohol intake either at baseline or followup. This evidence, in summary, indicates the potential importance of alcohol restriction in blood pressure control.

Role of Other Minerals in Hypertension

Potassium

One of the earliest observations of the association of dietary potassium with reduced blood pressure concluded that "potassium salt regularly produced a decline in blood pressure, while sodium salt just as regularly produced a rise" (Addison 1928). It was later observed that potassium lowered blood pressure in individuals with hypertension even when dietary salt intake remained high (McQuarrie, Thompson, and Anderson 1936). This protective effect has also been observed in rats (Meneely, Ball, and Youmans 1957). These inverse associations have been reviewed extensively (Meneely and Battarbee 1976; Tannen 1983; Prineas and Blackburn 1985).

Population studies have shown both a positive relationship between sodium intake and blood pressure and an inverse relationship between potassium intake and blood pressure (Maddocks 1967; Sinnott and Whyte 1973; Bulpitt et al. 1986). Although the Framingham study showed no relationship between urinary potassium levels and blood pressure (Dawber et al. 1967), potassium intake was more strongly correlated than any other dietary factor to blood pressure in a Hawaii study (Reed et al. 1985). In disease prevalence studies in Evans County, Georgia, hypertension was more strongly associated with lower potassium intake in black adults than in white adults with similar sodium intakes (Grim et al. 1980). In a Southern California cohort of 859 men and women ages 50 to 79 years, an inverse relationship was observed between dietary potassium, estimated by 24-

hour dietary recall at the baseline examination, and death from stroke, identified by examination of death certificates, in the 12-year followup (Khaw and Barrett-Connor 1987).

Intervention trials have attempted to lower blood pressure in hypertensive patients with the use of potassium supplements. The effect of 60 mEq of potassium supplementation was compared with that of placebo and was documented in a double-blind 4-week crossover trial in 23 patients with hypertension. A small but significant lowering of both systolic and diastolic blood pressure occurred in the supplementation period compared with the placebo period (MacGregor, Smith, et al. 1982). In another double-blind crossover trial, 20 normotensive subjects taking a 64 mEq of potassium supplement reduced diastolic blood pressure significantly (by 2.4 mm Hg) in 2 weeks compared with subjects taking a placebo (Khaw and Thom 1982). A double-blind clinical trial that enrolled 116 adults with mild hypertension for 8 weeks found a 3.4 mm Hg greater decrease in systolic blood pressure and a 1.8 mm Hg greater decrease in diastolic blood pressure in the group taking potassium supplements of 120 mEq/day than in the group taking placebo. A much greater decrease was observed in the five black patients taking potassium compared with placebo (Svetkey et al. 1987). In a 15-week clinical trial that randomized 37 mildly hypertensive patients, there was a mean difference (lower) of 14 mm Hg in systolic blood pressure and 10.5 mm Hg in diastolic blood pressure in the patients taking 48 mEq/day of potassium compared with placebo (Siani et al. 1987). These beneficial effects of potassium on blood pressure in hypertensive patients have been confirmed in another randomized blinded crossover study of 19 hypertensive patients with diuretic-induced potassium depletion. Potassium supplements of 60 mEq/day reduced mean blood pressures by 5.5 mm Hg (Kaplan, Carnegie, and Raskin 1985). In contrast to these findings, an unblinded crossover study of 12 mildly hypertensive subjects showed no influence of potassium supplementation on blood pressure (Richards et al. 1984). One extensive review of the literature concluded that a high potassium intake has no effect on blood pressure in normotensive animals or humans, although a potassium intake of 120 to 175 mEq/day appears to reduce blood pressure in hypertensive patients by 3 to 10 percent (Tannen 1983).

If potassium does have an effect, it is likely influenced by sodium. In a randomized 12-week crossover study of increased sodium intake and added potassium in hypertensive and normotensive adults, blood pressure rose in both hypertensive and normotensive subjects who consumed increased sodium, but among hypertensives who consumed supplemental potassium, blood pressure fell (Parfrey et al. 1981).

The relationship between sodium, potassium, and blood pressure in normotensive adults has been found to be dependent on family history of hypertension (Pietinen, Wong, and Altschul 1979). The response to dietary potassium and sodium has been assessed in male medical students with and without a family history of hypertension. In just the group with a positive family history, diastolic blood pressure was reduced by about 11 mm Hg when sodium intake was lowered in the presence of high potassium intake compared with 7 mm Hg with reduced sodium intake alone (Parfrey et al. 1981). A reanalysis of these data confirmed this effect of potassium (Holly et al. 1981). Also consistent with these data that show a lack of potassium influence in those with a negative family history of hypertension are two trials that failed to find a blood pressure-lowering effect of potassium in those persons who achieved substantial reduction of sodium intake (Morgan et al. 1983; Smith et al. 1985). Because the duration of the majority of these studies has been only weeks or months, longer term intervention studies have been recommended to evaluate the effects on blood pressure of both increased potassium and reduced sodium (Prineas and Blackburn 1985).

Calcium

A possible role for dietary calcium in the regulation of blood pressure is suggested by experimental studies in animals, epidemiologic studies, and clinical studies, including clinical trials (Kaplan and Meese 1986).

Experimental studies in spontaneously hypertensive rats have suggested that supplemental dietary calcium may prevent the development of hypertension (Ayachi 1979; McCarron, Henry, and Morris 1982; Schleiffer et al. 1984). One study, however, reported no such effect (Stern et al. 1984).

An association between calcium and blood pressure was suggested by reports that "hard" drinking water (containing calcium) was associated with reduced prevalence of cardiovascular disease (Schroeder 1960). This association between calcium and blood pressure is supported by observations from the Puerto Rico Heart Health Program that individuals who drank no milk (which is high in calcium) had twice the prevalence of hypertension as those who consumed a quart of milk or more per day (Garcia-Palmieri et al. 1984). An analysis of data for 5,050 adults in Southern California surveyed for heart disease risk factors as part of a Lipid Research Clinics population study indicated that hypertensive men, but not hypertensive women, had a significantly lower intake of calcium from milk than normotensive individuals; in men, diastolic blood pressure decreased significantly with reported increasing milk consumption (Ackley, Barrett-Connor, and Juarez 1983). In a case-control study, dietary calcium

intake assessed by 24-hour dietary recall in 46 hypertensive patients and normotensive controls showed that calcium intake was 22 percent less in the hypertensive group (McCarron, Morris, and Cole 1982).

An analysis of the National Health and Nutrition Examination Survey I (NHANES I) data found an inverse, although inconsistent, association between dietary calcium and blood pressure; the lower blood pressures were correlated with higher levels of serum calcium (Harlan et al. 1984). Although a subsequent examination of a subset of NHANES I data also suggested an inverse relationship between dietary calcium intake and blood pressure (McCarron et al. 1984), the analysis was hindered by methodological shortcomings: the salt added in food preparation and at the table were excluded from dietary intake data; the results were examined using only systolic blood pressure measures; and the analyses did not control consistently for age, race, sex, and body mass index (Feinleib, Lenfant, and Miller 1984). Subsequent analyses of both NHANES I and II data have failed to identify a relationship between dietary calcium and blood pressure except among black males in NHANES I (Sempos et al. 1986).

In a randomized placebo-controlled clinical trial, normotensive adults taking 1,000 mg of calcium per day for 8 weeks had blood pressures that were 6 to 9 percent lower than those receiving placebos (Belizan et al. 1983). In an uncontrolled clinical study, 2,000 mg of oral calcium carbonate taken for 5 days lowered blood pressure in people with low plasma renin activity (Resnick, Nicholson, and Laragh 1984). A blood pressure-lowering effect of calcium has been noted after 5 months in an uncontrolled study of individuals with mild-to-moderate hypertension (Resnick, Nicholson, and Laragh 1984).

Another randomized placebo-controlled crossover trial provided 1,000 mg/day of oral calcium for 8 weeks to 48 hypertensive and 32 normotensive individuals. In the hypertensive group, standing systolic and diastolic pressures were significantly reduced (by 6 and 2 mm Hg, respectively), as was supine systolic (by 3 mm Hg)—but not diastolic—pressure. Among normotensive persons, only supine diastolic blood pressure was significantly lowered with calcium (McCarron and Morris 1985). Supplements of 1,500 mg of calcium per day produced modest (2 to 3 mm Hg) reductions of systolic and diastolic pressures in both white and black normotensive men in a recent study (Lyle et al. 1987). An oral calcium supplement of 10 or 20 mM/day was given for 2 months in a double-blind randomized crossover study involving 51 hypertensive and 51 normotensive patients. There was an apparent trend for lower systolic blood pressure. The diastolic blood



pressure did not change significantly. In the normotensive group, there were no significant changes in blood pressure (Nowson and Morgan 1986). The effect of 1 g of calcium lactate gluconate or calcium carbonate in 23 patients with mild-to-moderate hypertension was observed for 8 weeks in another double-blind crossover study. These investigators reported no evidence that oral calcium supplements lowered blood pressure in these patients (Zoccali et al. 1986).

Analyzed collectively, the preceding evidence suggests that a disturbance in cellular calcium metabolism may contribute to the development of hypertension, although the physiologic mechanism for this effect is as yet unknown (Pak 1985). The possibility that calcium supplements may lower blood pressure only in patients with mildly elevated levels of parathyroid hormone that have occurred as a result of abnormally high kidney excretion of calcium (and consequent lowering of blood calcium levels) has been proposed on the basis of a study of 90 hypertensive patients (Grobbee and Hofman 1986b). At present, the role of calcium in blood pressure regulation must be considered uncertain and the clinical evidence considered inconclusive (Kaplan and Meese 1986).

Chloride

In early studies, the blood pressure-raising effect of sodium chloride was attributed to its chloride portion. Since the mid-1950's, however, the sodium component of sodium chloride typically has been considered more important (Dahl and Love 1954).

The effect of dietary chloride on the pathogenesis of sodium-dependent hypertension has been examined in experimental animals. Steroid-treated rats with one kidney removed developed hypertension when fed sodium chloride but not when they were fed sodium bicarbonate. These different responses could not be explained by differences in caloric intake, weight gain, or the balance of sodium, water, or potassium (Kurtz and Morris 1983).

The observation that salt-sensitive hypertension depends on the presence of both chloride and sodium has induced some investigators to suggest that chloride—rather than sodium—induces hypertension. In one study of salt-sensitive rats, for example, blood pressures were shown to be significantly higher after 5 weeks of loading with sodium chloride, but they remained normal when sodium bicarbonate was substituted for sodium chloride in the diet (Kotchen, Luke, and Ott 1983). The effect of a nonchloride sodium salt on blood pressure was recently tested in five hypertensive men. At 10 mEq/day of sodium chloride, blood pressure was normal. A 230 mEq of

sodium chloride supplement induced a significant increase in blood pressure, which was reversed with a supplement of an equimolar amount of sodium as sodium citrate (Kurtz, Al-Bander, and Morris 1987). This study again raises the possibility that the chloride ion may independently contribute to the sodium chloride-induced increases in blood pressure. The need for further investigation is reinforced by the report that chloride concentrations and activity are decreased in erythrocytes of humans with essential hypertension (Kurtz and Morris 1985).

Magnesium

An early study showed that clinical administration of magnesium salts lowered blood pressure (Blackfan and Hamilton 1925), and recent epidemiologic data have rekindled an interest in this effect (Joffres, Reed, and Yano 1987). Because magnesium, along with calcium, is present in "hard" drinking water, an association between dietary magnesium and blood pressure is also suggested by the finding of lower rates of cardiovascular disease in hard-water areas. The possible association between magnesium and blood pressure is supported to some extent by an observation that magnesium-deficient rats had both higher blood pressure levels and reduced diameter of tiny blood vessels than control animals (Altura et al. 1984).

Among elderly people in Denmark, low serum magnesium concentration has been associated with increased blood pressure (Petersen et al. 1977), and magnesium intakes have been found to be reduced among hypertensive patients (McCarron 1982). One randomized study of 18 patients taking diuretic medications for hypertension or congestive heart failure showed that magnesium supplementation was followed by a significant fall in both systolic (by 12 mm Hg) and diastolic (by 8 mm Hg) blood pressure (Dyckner and Webster 1983). As stated in the chapter on drug-nutrient interactions, thiazide diuretics have been associated with magnesium depletion, and this may be a factor in the blood pressure changes seen during magnesium supplementation. However, in another study in which patients with mild to moderate hypertension not on diuretics were given either magnesium supplements or a placebo, blood pressures did not fall in either group, despite a significant increase in plasma magnesium concentration and in urinary magnesium in the group taking the supplement (Cappuccio, Markandu, and Beynor 1985). The inconsistency of data regarding magnesium and blood pressure levels indicates that there are many unanswered questions regarding the role of magnesium in hypertension.

Trace Elements

Little is known about the role of trace elements in the regulation of blood pressure. Because trace elements such as zinc, copper, and iron participate in enzyme reactions related to blood pressure regulation, they could be factors in the development of hypertension, although they are unlikely to be its primary cause (Saltman 1983). In a comprehensive review of this topic, cadmium was the only trace element that appeared to be related to blood pressure (Mertz 1985), and experimental data from animals and the results of some human studies are consistent with a potential causative role for this element. This role, however, lacks confirmation and is complicated by the many interactions of cadmium with selenium, copper, zinc, and lead. Epidemiologic and animal data also suggest a direct relationship between lead levels and blood pressure (Pirkle et al. 1985), but existing data do not support a major role for this or any other trace element in the pathogenesis of hypertension.

Role of Macronutrients in Hypertension

Carbohydrates

There is some evidence that carbohydrates may play a role in blood pressure regulation. Studies in rats have indicated that dietary sucrose increases blood pressure, whereas starch decreases it (Ahrens et al. 1980). Normotensive humans who supplemented their diets with 200 g/day of sucrose had diastolic blood pressures 5 mm Hg higher than those who consumed no added sucrose (Ahrens 1974). Both a high sucrose and a high glucose solution—but not galactose or lactose—consumed after an overnight fast produce transient increases in blood pressure in normotensive men. These observations suggest that carbohydrates might influence blood pressure-regulating hormones in rats, but investigations conducted to date have failed to find a similar effect in humans (Hodges and Rebello 1985).

Fiber

There is some indication that plant fiber may reduce blood pressure levels (Anderson 1983; Anderson and Tietzen-Clark 1986), but it is uncertain whether dietary fiber plays a role in blood pressure regulation that is independent of other concomitant dietary changes, such as replacement of fat or facilitation of lower sodium intake (Mendeloff 1985).

Fat

The effect of dietary fats on blood pressure has been recently reviewed (Sacks et al. 1987). Neither the effect of total fat content nor responses to major changes in fatty acid intake have been sufficiently studied with desirable methodology, controlling potentially confounding variables for an adequate time period.

Polyunsaturated Fatty Acids. Considerable interest has focused on observational studies indicating that an increased intake of polyunsaturated fatty acids is associated with lower blood pressure (Iacono, Puska, and Dougherty 1983; Puska et al. 1985). Although it is uncertain whether the lower blood pressures were attributable to the change in the type of dietary fat, an action of the fatty acids on blood pressure might be mediated through changes in prostaglandin metabolism caused by increased intake of the polyunsaturated fatty acid, linoleic acid. Some prostaglandin metabolites influence salt and water excretion and can cause contraction or dilation of small blood vessels, thereby affecting blood pressure (Iacono et al. 1981). A single-blind randomized control experiment among hypertensive and normotensive men and women in North Karelia, Finland, found that a low-fat diet with a P/S ratio of 1.0 was associated with reductions in systolic and diastolic blood pressure of 8.9 and 7.6 mm Hg, respectively, reductions greater than those observed in parallel control groups receiving sodium restriction or no dietary change (Puska et al. 1983). Another team of investigators has shown that compared with the baseline diet, decreasing saturated fats and increasing polyunsaturated fats are associated with lower blood pressure (Iacono et al. 1975; Iacono et al. 1981; Iacono, Dougherty, and Puska 1982).

No significant change in blood pressure was observed after feeding either a low-fat diet (22 percent of calories from fat) or a diet high in polyunsaturated fatty acids (19 percent of calories from polyunsaturated fatty acids—compared with the U.S. average of about 7 percent) to 15 to 18 normotensive young adults (Brussaard et al. 1981). A double-blind randomized control trial of 6 weeks studying 21 mildly hypertensive patients found that increasing the polyunsaturated fatty acid linoleate from 4.6 to 13 percent of calories and reducing saturated fatty acids from 16 to 10 percent failed to produce any significant changes in blood pressure (Sacks et al. 1987).

Monounsaturated Fatty Acids. An association between increased consumption of the monounsaturate oleic acid and reductions in both systolic and diastolic blood pressures has been observed recently in a cross-sectional survey that examined 3-day food records and resting blood

pressure in 76 normotensive men. Although no physiologic explanation for this association is evident, it is consistent with the lower prevalence of hypertension among Mediterranean populations who consume diets rich in oleic acid-containing olive oil; Mediterranean populations also have a high carbohydrate intake and low saturated fatty acid intake compared with the U.S. population (Williams et al. 1987).

Omega-3 Fatty Acids. The role of omega-3 fatty acids in blood pressure lowering has also received attention. Investigators have reported that the ingestion of enough fish to provide 5 g of omega-3 fatty acids per day for 2 weeks caused a significant fall in blood pressure, and the lower blood pressure could be maintained subsequently by the weekly consumption of 20 oz of fish that provided 1.2 g/day of omega-3 fatty acids (Singer et al. 1986).

In summary, these results indicating that the type of fat intake may influence blood pressure level require further investigation to resolve inconsistencies, confirm the observations, and establish their clinical significance (Iacono, Dougherty, and Puska 1982).

Protein

The possibility of an effect of protein level on blood pressure regulation and whether specific amino acids might have an antihypertensive role has received little scientific study. In a study of spontaneously hypertensive rats, those rats fed American rat chow containing 25.3 percent protein, compared with rats fed Japanese rat chow containing 19.7 percent protein, developed a lower incidence of stroke (30 percent versus 80 percent). When the rats previously fed American rat chow were fed the Japanese diet, an accelerated rate of cerebral lesions and stroke occurred. This observation led to a search for specific amino acids in proteins that might affect blood pressure; tryptophan and tyrosine, as well as total protein intake, were cited as leading possibilities. Taurine has been demonstrated as having an antihypertensive effect in patients with hypertension (Kohashi et al. 1983) as well as in rats (Abe et al. 1987). One hypothesis is that amino acids or protein may affect blood pressure either at the vascular level or through changes in neuronal control of the cardiovascular system (Yamori et al. 1984), but this idea has yet to be confirmed.

Role of Caffeine in Hypertension

The consumption of 150 mg of caffeine (two to three cups of brewed coffee) may promote an increase in blood pressure by 5 to 15 mm Hg within 15

minutes that is maintained for as long as 2 hours. These short-term effects are primarily mediated by an increase in cardiac output, with systolic pressure usually rising more than diastolic; they are demonstrable both in individuals who do not habitually consume caffeine and in those who do habitually ingest caffeine if they abstain for 12 hours or more. The cause of these effects is uncertain; short-term effects are not directly attributable to rises in plasma catecholamines, vasopressin, or renin activity (Izzo et al. 1983).

Chronic caffeine consumption, however, neither maintains high blood pressure nor is it associated with increased rates of hypertension (Robertson et al. 1984). Prolonged administration of caffeine—as much as 504 mg/day for 4 weeks—has not been associated with significant rises in blood pressure either in normotensive (Ammon et al. 1983) or hypertensive individuals (Robertson et al. 1984). Thus, there appears to be adaptation or tolerance to the hemodynamic effects of caffeine.

Implications for Public Health Policy

Dietary Guidance

General Public

Dietary factors that clearly contribute to high blood pressure include obesity and excessive intake of sodium and alcohol. The average daily sodium consumption of 4 to 6 g by adult Americans is substantially above the National Research Council's recommended range of 1.1 to 3.3 g for safe and adequate intake and is 5 to 10 times higher than the amount required. Many individuals are able to maintain normal blood pressure levels over a large range of sodium intake; the lack of known harm from moderate sodium restriction, however, and the potential benefit to people whose blood pressures rise with increased sodium intake suggest that those who ingest excess sodium—most Americans—should consider reducing their dietary sodium intake.

The strong association between obesity and hypertension and the demonstrated reduction in blood pressure that occurs with weight loss suggest that maintenance of desirable body weight should be a goal for the population.

Similarly, there is a direct association between blood pressure and alcohol consumption beyond about two standard-sized drinks daily. (One standard-sized drink is defined as 12 oz of regular beer, 5 oz of wine, or 1½ oz of distilled spirits.)

Some evidence indicates that a reduction in blood pressure is associated with increased dietary intake of potassium, calcium, magnesium, and fiber. This evidence is, as yet, too preliminary to recommend increased intake of these factors for the general population for the purpose of hypertension control. Likewise, although increased intake of certain lipids (e.g., omega-6 or omega-3 polyunsaturated fatty acids) may decrease blood pressure, additional research is needed before any recommendations can be made.

Special Populations

Achieving and maintaining desirable body weight and moderating sodium and alcohol intake can lower blood pressure in patients with mild and moderate hypertension and reduce the need for antihypertensive medications. Such patients should be informed of the likely benefit of these dietary practices, along with the importance of adequate caloric expenditure through exercise, and the moderation of fat intake, especially saturated fatty acids, to reduce high blood cholesterol levels and the risk for heart attack.

Nutrition Programs and Services

Food Labels

Evidence related to the role of dietary factors in hypertension supports the need for nutrition labeling on a wide selection of foods so that the consumer has the option of choosing foods with known amounts of calories and sodium.

Food Services

Evidence related to the role of dietary factors in hypertension suggests that food service programs should provide adequate choices of foods that provide essential nutrients and energy to maintain desirable body weight and should include foods that are low in sodium.

Food Products

Evidence related to the role of dietary factors in hypertension suggests that food manufacturers should continue to reduce sodium in products and continue the research and development of products low in calories and sodium.

Special Populations

Counseling and assistance in the selection and preparation of foods low in sodium and calories and assistance with the development of dietary pat-



terns that control energy, sodium, and alcohol should be available to individuals whose blood pressure places them in the mild-to-moderate as well as high range of hypertension.

Research and Surveillance

Special priority is attached to the following research and surveillance tasks related to the role of diet in hypertension:

- Development of practical methods for the rapid and reliable identification of individuals at high risk for hypertension because they are salt sensitive.
- Investigation of the interactions of sodium with other nutrients—such as potassium, calcium, chloride, magnesium, fatty acids, and fiber—in influencing blood pressure.
- Investigation of the role of specific dietary factors, including potassium, calcium, fatty acids, fiber, amino acids, trace elements, and alcohol, in the cause and potential prevention of hypertension and the mechanisms for these effects.
- Investigation of the mechanism of obesity-associated hypertension and determination of the ratio of fat to lean body mass that might prevent development of hypertension.

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